pies are often initiated together, it is often difficult to decide if there is more benefit associated with one form of treatment or another.

**With regard to CKD-MBD, what is your treatment approach to patients with stage III–IV CKD and stage V CKD?**

Inasmuch as abnormalities in bone and mineral metabolism begin early in the course of CKD, it is desirable to initiate treatment as early as the course of kidney disease possible. Current practice recommendations are to screen for abnormalities in bone and mineral metabolism by the early measurement of intact PTH and, if PTH is elevated, to consider initiating treatment.

The initial step in treatment would be to measure serum calcium and vitamin D and, if they are low, to try to bring them into the normal range. If this is not sufficient to control the hyperparathyroidism, beginning an active form of vitamin D, such as calcitriol, paricalcitol, or doxercalciferol, should be considered. Although studies in experimental animals have shown the efficacy of phosphate restriction even in the early stages of CKD, one could consider efforts to decrease dietary phosphate intake or consider the use of phosphate binders, although these are not yet approved for use in early CKD.

As kidney disease advances, the need for active forms of vitamin D becomes more common, and phosphate binders are indicated when hyperphosphatemia occurs. In stage V CKD, or in patients receiving dialysis, the initial approach is to control phosphate first, then to correct hypocalcemia, to use active vitamin D sterols, and if this is insufficient to control PTH to target values, then to consider the use of a calcimimetic agent. In terms of the types of phosphate binders used, one has to be cognizant of the existence of vascular calcification and the risk of its progression and to consider limiting the ingestion of calcium-containing phosphate binders. Although this approach is somewhat controversial at present, it appears to be a prudent one, especially in the presence of existing vascular calcification.

**When do you usually start calcimimetic therapy?**

In patients receiving dialysis, it is our practice to try to control hyperparathyroidism with phosphate control and active vitamin D sterols. As you know, there is a broad spectrum of patients, some who are hypercalcemic and some of whom have serum calcium values at the upper limits of normal. The former are easily treated with active vitamin D sterols, whereas in the latter, the use of active vitamin D sterols may be limited by a tendency to hypercalcemia. If hyperparathyroidism cannot be controlled to target values, then the addition of a calcimimetic agent is indicated. It is our practice to use this in addition to the other measures.

**What changes can be expected with CKD-MBD after successful kidney transplantation?**

In general, the abnormalities associated with CKD-MBD begin to improve after successful kidney transplantation. The abnormalities may be influenced by concomitant therapy and, indeed, by a previous history of CKD, such that many patients come to transplantation with adynamic bone disease, which may be the result of the time spent receiving hemodialysis, or indeed with complicating factors, such as prior steroid therapy or postmenopausal osteopenia. Further, transplantation is associated with bone loss, and it should be assessed in terms of fracture risk and treatment undertaken if necessary. The extent of the improvement in CKD-MBD after transplantation will, of course, vary according to the level of function achieved.

**What are the treatment options for osteoporosis with CKD? Please discuss bisphosphonates, PTH, and denosumab.**

Osteoporosis and other causes of low bone density often occur in patients with CKD throughout the entire spectrum of patients seen. Many of the agents used for the treatment of osteoporosis, such as bisphosphonates, are problematic, mainly because of a scarcity of studies, the long half-life of bisphosphonates in bone, and the possible presence of adynamic bone disease. In general, a precise diagnosis should be made before therapy is initiated. In recent years, other options for the treatment of osteoporosis have included intermittent injections of PTH, and there are a few data to support this approach in patients with CKD. The most recent therapy with denosumab has not been studied in detail in osteoporosis associated with CKD, but it may be a reasonable option, at least in some patients.

**What criteria do you use to recommend patients for parathyroidectomy?**

Parathyroidectomy should be considered for patients who have severe hyperparathyroidism that cannot be controlled by medical means. The failure to control hyperparathyroidism may be from inability to control serum phosphorus or persistent hypercalcemia, which limits the use of active vitamin D. Therapy with calcium and calcimimetics may limit the use of calcimimetic agents. Then, parathyroidectomy should be considered.

**Do you have any practice pointers for our readers?**

The most important practice pointer at present is to consider the treatment of abnormalities in bone and mineral metabolism early in the course of CKD and to begin measures in an attempt to limit the severity of the problem. In patients with advanced CKD, consideration of associated comorbidities may be useful in deciding on a particular approach or combination of approaches to treatment.

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**New Agents Hold Promise in Diabetes Treatment**

Sodium-dependent glucose co-transporter 2 (SGLT2) inhibitors and bile acid sequestrants hold promise for the treatment of type 2 diabetes. Both were among the agents highlighted in the symposium, “Novel Therapies for Type 2 Diabetes– Today and Tomorrow,” at the American Diabetes Association annual meeting in San Diego.

SGLT2 inhibitors, eight of which are in clinical trials, are the first class of drugs to target renal glucose reabsorption for treating diabetes, said Ernest M. Wright, PhD, DSc, of the University of Michigan Medical School. Studies have shown that activating PPAR-γ may help prevent diabetes nephropathy by blocking the effects of glucose and renin-angiotensin-aldosterone system (RAAS) in triggering podocyte apoptosis.

Researchers’ interest in the PPARs, however, is accompanied by substantial caution owing to the side-effects associated with the agents. They include bone fractures, fluid retention, weight gain as well as bladder cancer and cardiovascular risk.

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**Study participants**

Our study participants were on dialysis and had a mean age of 60 years. They were divided into two groups: one received placebo and the other received the SGLT2 inhibitor, empagliflozin.

**Study results**

After 12 weeks, participants in the empagliflozin group showed a significant decrease in body weight and blood pressure compared to the placebo group. They also exhibited a significant reduction in HbA1c levels, indicating improved glycemic control.

**Conclusion**

SGLT2 inhibitors offer promising potential as a new class of antidiabetic drugs. Further studies are needed to fully understand their therapeutic effects and side-effects in the context of diabetes management.

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**Practice Pointers continued**